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Editorial

The Role of the Orthopedic Surgeon in Preventing Low Back Pain Chronification

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Low back pain is one of the main causes of disability. The orthopedic surgeon is usually the first medical assistant in many countries or even the professional who will manage the patient subsequently.

Eventually low back pain is only an expression of another clinical pathology or an expression of a mental health disorder. Many non-orthopedic factors are enrolled to its manifestation or its contribution.

Most orthopedic surgeons are unable to adequately address either for lack of specific training either by they are not used to manage these patients. Even the most experienced experts in chronic pain management would not manage effectively some of these cases. What factors would be responsible for these heterogeneous clinical manifestations?

Every orthopedic surgeon ought to know of the existence of the factors that would interfere in the chronification of episodes of acute low back pain. By identifying such factors, this professional could manage or forward these patients to other professionals who could help on their assistance.

Pathophysiology of pain

Pain acts as an adaptation and protection mechanism. Painful stimuli lead to the emanation of behaviors and reflex responses that would promote the withdrawal of the triggering stimulus and, thus, prevent the worsening its damage.

The pathway of nociceptive impulse occurs ascendingly to the brain and locally at each level of the central nervous system. There are some alternative pathways of feedback that regulate the transmission of impulses, which can inhibit or amplify them.

Mechanical, thermal or chemical stimuli of peripheral nociceptors promote a potential from the afferent fiber to the dorsal horn of the spinal cord [1]. The axons then transmit the stimulus to medullary neurons, which are divided into local interneurons, propriospinal neurons and projection neurons. Projection neurons send long ascending fibers to the thalamus and brainstem [2].

Both at the medullary level and in the corresponding nuclei in the brainstem, local interneurons act as modulators with excitatory or inhibitory capacities [3]. Basically, what differentiates them are presynaptic neurotransmitters, GABA and endogenous opioids being inhibitory while substance P and glutamate being excitatory [4].

Supramedullary neurons transmit ascending information through two main pathways [5]. The lateral pathway is related to the sensory and discriminative component of pain, which provides information on the intensity, location and type of the nociceptive stimulus. In the other hand, the medial pathway is related to the affective and cognitive component, in constant transformation overtime.

In episodes of prolonged pain, there are adaptive and reversible phenomena of peripheral and central sensitization of the nociceptive pathways. They are initially physiological responses in order to protect a tissue with actual or potential damage [6]. However, the sustained continuation of the stimuli may be associated with the development of pathologic responses.

In pathological responses, there are a predominance of excitatory modulation [7-10]. The amount and arrangement of the inhibitory and excitatory fibers changes overtime, i.e. they undergo neuroplasticity. The impairment caused by neural hyperexcitation and by the failure of the descending inhibitory pathway contributes to pain chronification.

This process of inhibition or excitation can be evaluated through conditioned pain modulation (CPM). This method has been shown to be of great value in predicting the risk of chronifying a pain syndrome, as well as predicting the response to certain medications. The growing interest in CPM can also be by giving reproducible numbers to a subjective symptom, minimizing the influences of possible simulations.

Several authors have identified a lower-than-expected CPM response when compared to the control group in people with osteoarthritis of the knee [11,12], fibromyalgia [13,14], irritable bowel syndrome [15], temporomandibular disorder [16], migraine, and tension-type headache [17]. In 70% of people with chronic pain there is no inhibitory response to the test [18].

Temporal summation reflects hyperexcitation of the dorsal horn of the spinal cord and supramedullary interneurons. Painful perception increases progressively. Also, it is possible to identify such effect in clinical practice. Patients become more sensitive to repetitive stimuli, suggesting that the mechanisms that support chronic disorders may be involved in the onset or maintenance of clinical pain [19].

The spatial summation is a mechanism of sensorial hyperstimulation from the convergence of multiple afferents to common nociceptive pathways, ultimately triggering a sensation of pain following innocuous stimulation or amplifying pain after painful stimulation [20]. The sum of excitatory synaptic potentials increases the likelihood of reaching the presynaptic threshold, culminating with an action potential.

Psychological and social factors

The nociceptive stimuli interact with the emotion, decision making, and communicative tendencies. These factors depend on each person's cognitive ability as well as their momentary needs. Finally, the product of this complex combination is a subjective experience influenced by earlier, current and environmental experiences.

The interrelation of new sensory stimuli with previous experiences incites another set of behaviors modulated by feelings and intimate needs. Along the course of a lifetime, each of the factors cited has a different weight from previous ones, therefore it results in a new behavior.

Initially protective behaviors may acquire conscious or unconscious meanings related to subsistence and depend on volatile social factors, previous traumas, interaction with the environment, beliefs, virtues and values, needs, and momentary priorities.

Psychosocial factors such as labor dissatisfaction and poor coping skills are significantly related to worse recoveries, however the variance of outcomes can be explained between 29% and 46% [18]. The interconnection of previously sensitized neurons may precipitate a new crisis of pain in situations of anxiety or psychological stress.

As the description of pain depends fundamentally on the reports of those who feel it, it becomes a tool to influence the environment and the response of others in the best perceived interest by each one [21,22].

Conclusion

Musculoskeletal degenerative disorders usually occur concomitantly with low back pain. Intermittent crises of exacerbation have different presentations overtime, despite the apparent stability of causal factors. This constant change of pain patterns generates more anguish to those who experience it.

Often, orthopedic surgeons observe patients with signs and symptoms suggestive of central sensitization from anamnesis and physical examination. Symptoms such as primary hyperalgesia, secondary hyperalgesia, allodynia and spontaneous pain suggest the presence of central sensitization.

Neglecting the central sensitization concomitant to orthopedic pathologies results in persistence of pain even after well-executed surgical procedures. Therefore, the identification of patients with the highest risk of chronification is possible and desirable. The multimodal adjuvant treatment to orthopedic procedures obtains better outcomes.

Assistant physician and patient should understand the pathophysiology of pain as well as the factors that facilitate and inhibit its manifestation. Patients who participate in the pain control process adhere more easily to the proposed treatments. In addition, we can reduce anxiety related to unrealistic expectations.

Besides psychosocial factors, which precede the painful manifestation and the orthopedic evaluation in several years, other factors related to low back pain chronification originate from the poor pain management in its acute phase. In other words, a negligent physician is partially responsible for the transition from a physiological to a pathological pain response.

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